

Tuberculous meningitis

BCG vaccination and severity of childhood tuberculous meningitis

V Rajshekhar

BCG vaccination may lessen the severity of, rather than completely prevent, serious forms of tuberculosis

In this issue of the journal (*see pages 1550–4*), Kumar *et al* report on the efficacy of bacillus Calmette Guérin (BCG) vaccination in favourably altering the course and outcome of tuberculous meningitis (TBM) in children. The value of BCG vaccination for preventing tuberculosis has been debated since its introduction nearly eight decades ago. Several trials, cohort studies, and case control studies have failed to provide a definitive answer to this contentious issue. A recent meta-analysis revealed that the protective effect of BCG vaccination in infants was only around 50% against all forms of tuberculosis.¹ It has been suggested that although BCG vaccination is not highly effective in preventing the illness, it does play a role in reducing the severity of the more serious forms of the disease such as TBM.² The authors of the present report set out to study this premise and also to document the presentation of the disease in vaccinated children. The strength of their work lies in the prospective collection of their data. The

authors' data suggest that children who had been vaccinated had a milder form of the disease and consequently had a better short term outcome than those who had not received vaccination. Vaccinated children also had fewer focal neurological deficits and higher cerebrospinal fluid (CSF) cell counts. The authors speculate that vaccinated children are probably capable of mounting a better immunological response than unvaccinated children (reflected in the higher CSF cell counts) and that this is responsible for the better outcome. It is, however, unclear from the authors' data whether there were factors other than vaccination that contributed to the severity of the illness in the unvaccinated children. It is pertinent to note that the duration of the disease at presentation to the hospital in unvaccinated children was on an average 10 days longer than that in vaccinated children. Although this difference was not statistically significant, it suggests that parents of unvaccinated children tended to ignore early symptoms of the

disease, thereby allowing the disease to progress in severity. This could be a reflection of the poorer socioeconomic status and lower educational background of the family which in turn could adversely affect the severity of the disease and its outcome. A more comprehensive and in depth study of the socioeconomic parameters of the families of the children, the nutritional status of the children, and a longer follow up period (the outcome was studied at discharge from hospital) would have strengthened this paper. These deficiencies apart, this study adds to the body of literature that indicates that the utility of BCG vaccination lies more in influencing the severity of the serious forms of the disease, such as TBM, than in providing absolute protection against the disease. In doing so, it supports the continuation of BCG vaccination as a part of the Universal Immunization Programme (UIP) in endemic countries such as India.

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Trigeminal neuralgia

Can MRI distinguish injurious from innocuous trigeminal neurovascular contact?

W P Cheshire

It may be better not to operate on patients with chronic facial pain

Traditional wisdom teaches that "good surgeons know how to operate, better ones when to operate, and the best when not to operate".¹ When treating patients with chronic facial pain, prudent application of this aphorism draws not only from experience

but also from rigorous scientific investigation. Lang *et al* (*pages 1506–9 of this issue*) have strengthened the base of scientific evidence that informs the clinical decision not to recommend microvascular decompression for persistent idiopathic facial pain (PIFP).

Suboccipital microvascular decompression of the trigeminal nerve root is well established for the treatment of trigeminal neuralgia. Postoperatively, immediate relief ranges from 91% to 97% and long term efficacy from 53% to 70%, with an estimated annual recurrence rate of 3.5%.² Of the treatments effective for alleviating the pain of trigeminal neuralgia, microvascular decompression uniquely addresses the underlying pathology, which is generally presumed to arise from vascular compression of the trigeminal nerve root entry zone. Redundant turns in senescent blood vessels commonly abut the trigeminal nerve root. Neurovascular contact may progress, in the susceptible patient, to pulsatile indentation, focal demyelination, aberrant discharges, and ephaptic neural transmission. These disturbances culminate in paroxysmal crescendos, particularly if nociceptive

inhibition is inadequate. Lancinating or electrical pain strikes fleetingly and repetitively in response to normally non-painful afferent stimuli, such as oral movement or light touch at a remote facial trigger zone.

But then, not all facial pains behave as trigeminal neuralgia, and craniotomy is not without risks. Although no controlled clinical trial has shown benefit from microvascular decompression in atypical, non-paroxysmal trigeminal distribution pain,²⁻⁴ microvascular decompression is occasionally tried for the individual patient with terrible intractable unexplained pain. Severely affected patients consult many physicians and may be willing to undergo invasive procedures of uncertain benefit.³ In such cases, magnetic resonance imaging (MRI) may suggest a rationale for surgical intervention should it happen to disclose a vessel coursing alongside the trigeminal nerve root. Recent advances in MRI spatial resolution have brought into view more of these neuro-

vascular liaisons. Finer imaging detail, however, alone cannot distinguish the pathological from the incidental. Systematic clinical correlations are required for the sake of diagnostic clarity and treatment validity.

Lang *et al* utilised highly sophisticated MRI with 3D reconstruction to assess 12 subjects with PIFP. Radiologists blinded to the laterality of the pain evaluated the images for neurovascular contact, which was frequent, occurring in nine subjects. Of distinct interest is the fact that the presence of neurovascular contact did not differ between the symptomatic and asymptomatic sides, discounting any causal relationship to pain. None of the PIFP patients had morphologies of grooving, distortion, or deviation of the trigeminal root, which are considered more specific for trigeminal neuralgia.

Visualisation of neurovascular contact, it must be concluded, is a non-specific finding that should not itself be used as a convenient way of establishing a

diagnosis or opting for surgery in the patient with PIFP. Satisfactory outcomes for patients are still best guided by clinical criteria, in particular, the temporal pattern, pain characteristics, and triggering factors.

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